

Neth Heart J (2015) 23:613–615
DOI 10.1007/s12471-015-0758-6

RHYTHM PUZZLE ANSWER



An unexpected ECG finding

Mathijs Kuiper · Albert Willems · Arthur A.M. Wilde

Published online: 8 October 2015

© The Author(s) 2015. This article is published with open access at Springerlink.com

Answer

The ECG in Fig. 1 shows a sinus rhythm of 90 bpm with an intermediate axis, PQ delay of 202 ms and a narrow QRS complex. Convex ST elevation is seen in the right precordial leads. The ECG could be considered suspicious for acute septal myocardial infarction. However, reciprocal ST-segment changes are lacking and the QRS complex does not show any suspect abnormalities either. The ECG also has aspects of hyperkalaemia; in particular the sharp high voltage T waves point in that direction. The ECG in Fig. 1 is also compatible with a type 1 Brugada pattern. The patient has never had any cardiac symptoms, nor a family history of acute cardiac death.

The patient was admitted to the intensive care unit for rhythm monitoring. Hyperkalaemia was based on the use of high-dose spironolactone, in combination with dehydration and accordingly matched the patient's comorbidity. Figure 2 shows the ECG taken after correction of the hyperkalaemia (5.2 mmol/l). This ECG returned to normal in a period of 4 h,

without ST elevation and no signs of Brugada. Considering this short period of time to normalisation, there is a strong possibility that high potassium levels are provoking this Brugada pattern, as has been described before [1, 2]. Subsequently, Brugada syndrome was confirmed by positive ajmaline provocation testing. Shortly after administration of ajmaline the exact same type of Brugada was reproduced (with alternative placement of leads V3 and V5 to the intercostal space above V1 and V2, respectively; Fig. 3). This drug-induced Brugada syndrome is considered to be a type with a low risk of acute cardiac death [3]. However, in general it is recommended to prevent fever and if fever occurs to perform an ECG for potential rhythm monitoring, and to avoid certain drugs with the potential to prolong the QT interval.

Conclusion

Hyperkalaemia-induced Brugada syndrome.

Conflict of interest None of the authors have any conflict of interest related to this report.

Open Access This article is distributed under the terms of the Creative Commons Attribution License which permits any use, distribution, and reproduction in any medium, provided the original author(s) and the source are credited.

References

1. Littmann L, Monroe MH, Taylor L 3rd, Brearley WD Jr. The hyperkalaemic Brugada sign. *J Electrocardiol.* 2007;40:53–9.
2. Postema PG, Vlaar AP, DeVries JH, Tan HL. Familial Brugada syndrome uncovered by hyperkalaemic diabetic ketoacidosis. *Europace.* 2011;13:1509–10.
3. Mizusawa Y, Wilde AAM. Arrhythmogenic disorders of genetic origin: brugada Syndrome. *Circ Arrhythm Electrophysiol.* 2012;5:606–16.

M. Kuiper (✉)

Department of Internal Medicine, Sint Lucas Andreas Hospital,
Amsterdam, The Netherlands
e-mail: mathijskuiper@hotmail.com

M. Kuiper

Department of Cardiology, Haga Teaching Hospital,
The Hague, The Netherlands

A. Willems

Department of Cardiology, Sint Lucas Andreas Hospital,
Amsterdam, The Netherlands

A.A.M. Wilde

Department of Cardiology, Academic Medical Centre,
Amsterdam, The Netherlands



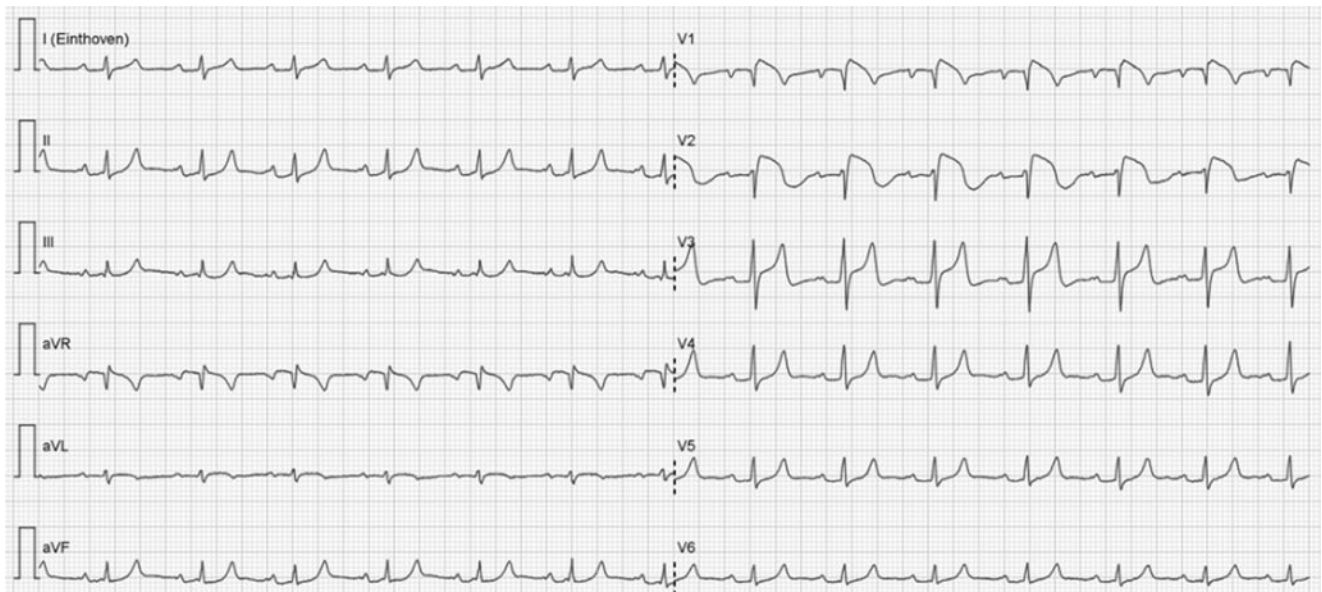


Fig. 1 ECG at presentation

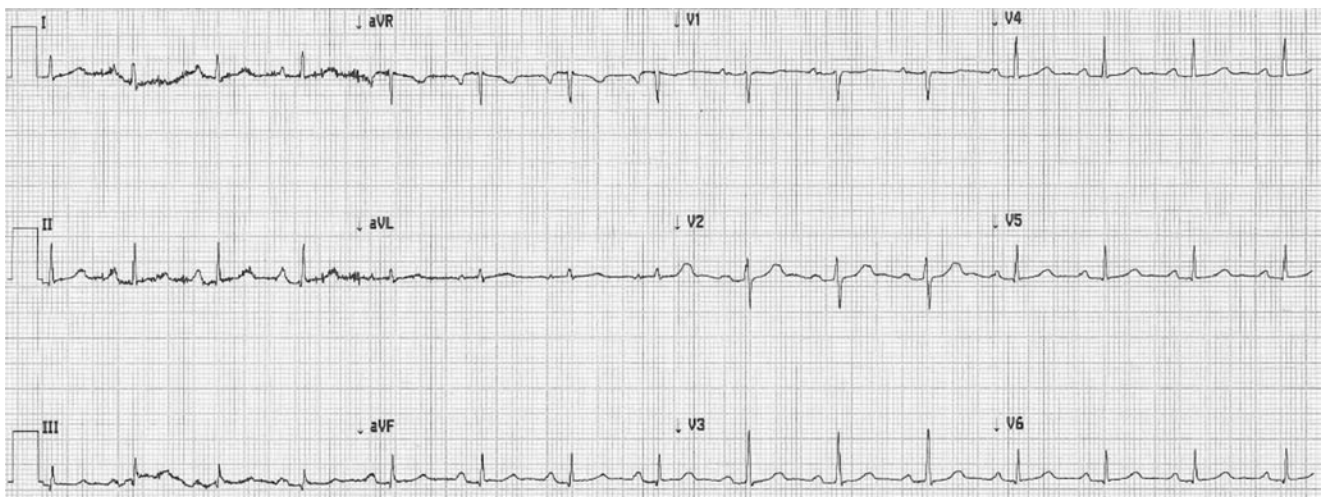


Fig. 2 ECG after correction of hyperkalaemia

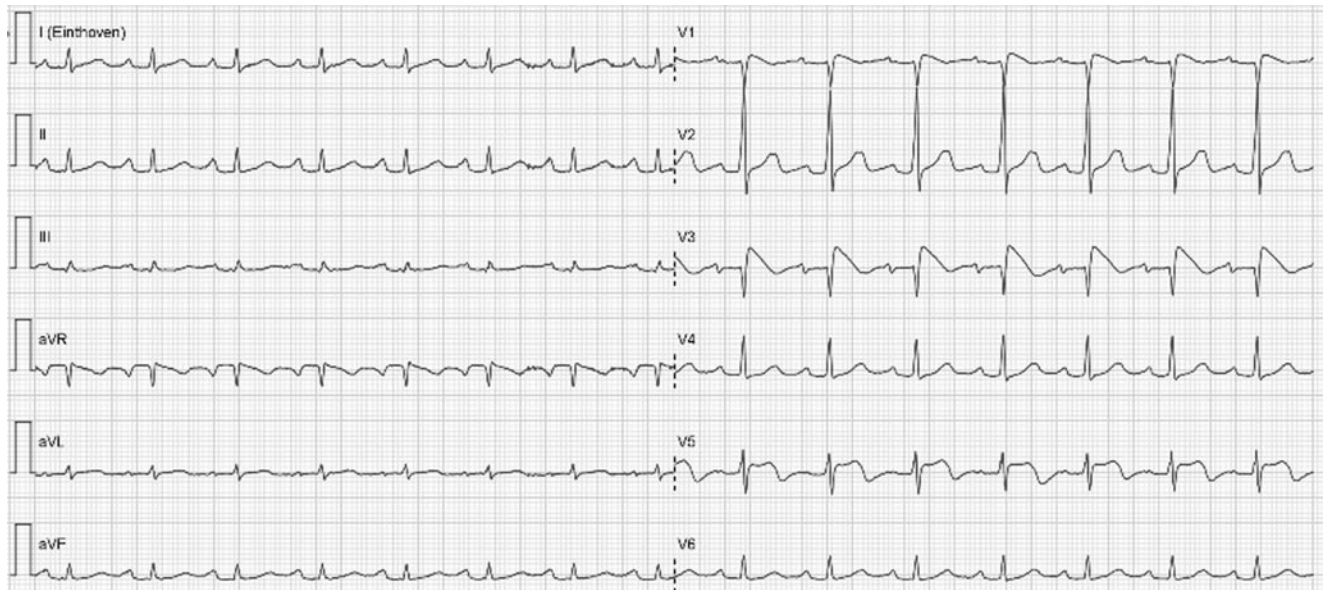


Fig. 3 ECG during ajmaline provocation testing

